Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services
Emergency Programs

Number 17-3 FALL 1989

This Issue

Emergency Field Investigations

Foreign Animal Disease Investigations. During the third quarter of fiscal year 1989 (April, May, and June), United States Department of Agriculture (USDA), Animal and Plant Health Inspection Services (APHIS) veterinarians conducted 60 investigations of suspected foreign animal diseases to eliminate the possibility that exotic diseases were introduced into the United States. Of the total, 15 were for suspected vesicular conditions; 20 for exotic Newcastle disease, avian influenza, and other poultry diseases; 2 for septicemias; 1 for encephalitis; 1 for mucosal disease; and 21 for exotic ectoparasites and miscellaneous conditions. Exotic Newcastle disease was found in pet birds on two premises in Connecticut and one in California. The California birds allegedly were smuggled. The birds were seized and placed in a USDA quarantine facility where it was determined that they were infected. The contaminated premises and facilities were cleaned and disinfected. As a precaution, a total of 119 pet birds on the affected California premises which had no known contact with the infected birds were tested for possible Newcastle disease virus, kept under quarantine, and retested. Results of the tests were negative.

Also, exotic ticks were found in Texas and Ohio on adult ostriches recently imported from Africa (see article below: Exotic ticks on Imported Ostriches).

No exotic diseases were found in livestock or poultry from a total of 146 investigations completed during the first 9 months of the fiscal year. It is the responsibility of each Foreign Animal Disease Diagnostician (FAD) to report all official investigations of suspected foreign diseases.

An **unidentified swine disease** has been reported in Indiana, primarily in swine kept in farrowing houses; manifested primarily in abortions, birth of dead and weak pigs, and mortality in neonatal pigs. Minor respiratory problems have been observed in adult and growing swine, including some that have nearly reached market weight. Broad spectrum antibiotic therapy reportedly has been beneficial in swine older than 3 weeks of age. The problem in a herd usually seems to subside 6 to 10 weeks after the first signs of disease are noticed. Subsequent farrowings appear to be normal.

Diagnostic samples have been submitted to the National Veterinary Services Laboratories (NVSL) and the veterinary diagnostic laboratories at Kansas State

University and Purdue University. An infectious cause is suspected; none has been found. Chlamydia, encephalomyocarditis virus, and influenza virus are among the suspected disease agents. The disease is thought to be either new or a newly recognized form of an established disease. Dr. Garry D. Weybright (APHIS Veterinary Medical Officer, P.O. Box 688, Monon, Indiana, 47959. Telephone number; Area Code (219) 253-7950) may be contacted for additional information. (Dr. M. A. Mixson and Dr. J. L. Williams, USDA, APHIS, Hyattsville, Maryland 20782, (301) 436-8073)

Foreign Animal Disease Update

The Office International des Epizooties (OIE) reported the following diseases during this quarter of the year (January, February, and March 1989):

In South America, Bolivia reported 50 outbreaks of **foot-and-mouth disease** (FMD). The virus was not typed. Paraguay reported one outbreak. Colombia reported 49 outbreaks of type O and 32 outbreaks of type A. Venezuela reported three outbreaks of FMD type A. Uruguay reported 2 outbreaks, involving 113 cases of FMD type O. The Pan-American Health Organization (PAHO) reported that Brazil had 54 outbreaks of FMD in January, and Argentina had 18 outbreaks in January and 10 in February.

In Europe, Italy reported two outbreaks of FMD type C, during March.

In the Middle East, Turkey reported 12 outbreaks of FMD type O. Israel reported one outbreak of FMD type O. Saudi Arabia reported FMD type O. Bahrain reported one outbreak of FMD type O.

In Asia, the Far East, and Oceania; Hong Kong reported four outbreaks of FMD. Pakistan had one outbreak of FMD, type unknown. Kuwait reported 15 outbreaks of FMD type O, with 143 cases.

Vesicular Stomatitis (VS). The Pan-American FMD Center reported that Colombia had 34 outbreaks of VS in February; 12 were caused by VS New Jersey virus, and 22 were caused by VS Indiana virus.

No outbreaks of **swine vesicular disease** (SVD), **rinderpest** (RP), **peste des petits ruminants** (pest of small ruminants), **Teschen disease** (TD), fowl plague (FP), or **Rift Valley fever** (RVF) were reported during the quarter.

The Congo reported outbreaks of **lumpy skin disease** (LSD), but the number was not given. Senegal reported an outbreak of LSD. Zimbabwe reported an outbreak in January with three cases and an outbreak in February with two cases.

Morocco reported 34 outbreaks of **sheep and goat pox** (SGP) since November 1988. Turkey reported 44 outbreaks of SGP in January and 65 in February. Kuwait reported 17 outbreaks in December with 111 cases, and 7 outbreaks in February with 12 cases. Pakistan also reported outbreaks of SGP.

South Africa reported 7 outbreaks of **African horse sickness** (AHS) in January and 30 outbreaks in February.

Italy reported one outbreak of **African swine fever** (ASF) in January and two in February. Malawi reported one outbreak in January. Namibia reported one outbreak. Portugal reported 36 outbreaks in January and 39 in February. The February

outbreaks in Namibia involved 214 cases. Senegal reported one outbreak in January. Spain reported 5 outbreaks of ASF in January and 26 in February.

Nine countries reported outbreaks of **hog cholera** (HC) during the quarter. In Austria, 6 outbreaks were reported involving 72 cases. Korea reported 2 outbreaks in January and 18 outbreaks in February. Hong Kong reported two outbreaks in January. Italy reported two outbreaks of HC and Malaysia reported one outbreak. Mexico reported 2 outbreaks in January and 12 in February involving 2,286 cases. Paraguay reported 1 outbreak with 20 cases. Taiwan reported 4 outbreaks with 372 cases. Uruguay reported 1 outbreak of HC with 50 cases.

Twelve countries worldwide reported outbreaks of **Newcastle disease** (ND) (untyped, presumed to be velogenic, viscerotropic type). ND was reported from Africa in Egypt, the Congo, Malawi, Botswana, and South Africa. In the Americas, ND was also reported from Mexico, Haiti, and Colombia. Haiti had 8 outbreaks involving 25,450 cases. ND was reported in Europe from Cyprus, Turkey, and Yugoslavia. In the area of Asia, the Far East and Oceania, untyped ND was reported from Korea.

Velogenic viscerotropic Newcastle disease (VVND) was reported from Botswana, Malaysia, and Pakistan.

Information that was reported during January, February, and March may include previously unreported data from outbreaks which occurred in previous months. In some instances, diagnosis may be strictly clinicopathologic without laboratory confirmation.

Necrotic hepatitis or viral hemorrhagic disease of rabbits reported in the Spring and Summer 1989 issues of the Foreign Animal Disease Report (17-1:8 and 17-2:7) as widespread in Europe and some Asian countries, was first reported in the Western Hemisphere in Mexico during December 1988. During the ensuing months, a total of 77,337 rabbits died of the disease in Mexico, and 42,747 were sacrificed in a disease eradication campaign carried out by the Mexican National System for Emergencies in Animal Health (SINESA), Secretariat of Agriculture and Water Resources (SARH). SINESA performed 7,012 investigations of suspected viral hemorrhagic disease in 10 States and the Federal District. By July 7, 1989, the eradication campaign was nearly complete. No new occurrences were reported after May 1989, and a total of 10 foci remained under investigation. (Dr. M. J. Gilsdorf, USDA, APHIS, International Services, Hyattsville, Maryland 20782, (301) 436-8892)

Errata

(Editor's note: In the Summer 1989 issue, 17-2:3, column headings for the following table were erroneously transposed. The corrected table is included in this Fall issue, to summarize data covering January, February, and March 1989).

Vesicular stomatitis (VS), types New Jersey (NJ) and Indiana (IND), was reported from Mexico, Panama, and Central and South America, for January, February, and March 1989. The following VS outbreaks were reported for the indicated country *.

	NJ	IND		NJ	IND
Mexico	1	1	Guatemala	50	10
Honduras	13	1	El Salvador	5	3
Nicaragua	11	0	Costa Rica	10	0
Panama	0	1	Colombia	42	36
Ecuador	2	0	Venezuela	1	0

*Data from Mexico, Central America, and Panama were reported to the Pan-American FMD Center. Data from South America were reported to the OIE.

Avian Salmonellosis

Clinical presentation of avian salmonellosis caused by *Salmonella enteritidis* (SE) phage-type 4 in poultry flocks may go undetected by the producer without attention to subtle signs (see 17-2:1 and 16-4:2-3). The organism can affect both broiler and layer-type flocks. Infected poultry may be in poor body condition or be moribund. The clinical picture is characterized by an increased mortality rate of up to 10 percent in week-old chicks. Uneven growth rate and stunting is evident in the flock by 3 to 5 weeks of age. The most common lesion on necropsy at this age is a retained yolk sac. The indurated yolk sac often can be palpated through the abdominal wall. Early signs of pericarditis may be detected. Necrotic foci and petechiae may be present on the liver.

In affected flocks, up to 5 percent of the poultry do not grow normally and appear as culls (poultry that are judged to be unprofitable and are removed from flocks). Mortality can reach 20 percent by the time of slaughter. Evidence of septicemia is often present in hens at slaughter. One reported sequel is pericarditis in broilers or pullets. The pericardial sac is grossly distended with up to 15 ml of fibrinous exudate which resembles mastitic milk. The pericardium is thickened and leathery, containing deeply congested blood vessels. Histologic changes in the myocardium and pericardium are compatible with a chronic inflammatory reaction. Pericarditis may be accompanied by other signs, including perihepatitis and air sacculitis.

Ovarian infection is present in roughly half of the affected hens. Congested and misshapen ovules and egg peritonitis are present in severe cases. The causative organism has been cultured from the eggs of hens with ovarian infection; however, egg culture is not considered a sensitive test. As with pullorum disease, the number of affected ovules may vary. Normal ovules can occur on the same ovary with diseased ones. Recovery rates for the organism on culture are reported to rank from highest to lowest as follows: environmental samples, fecal samples, gut (gastrointestinal tract tissues), dead embryos, internal organs, and ovaries.

Several variables influence the prevalence of disease on a premises. Biosecurity measures can confine the disease to a small number of houses. Intermittent shedding of SE from parent and grandparent flocks has been reported to limit disease to one house on a premise, despite common origin of stock. The primary mode of transmission (transovarian versus horizontal) appears to influence prevalence and distribution within a flock. The relative importance of these modes is not clear. (Dr. Sean F. Altekruse, USDA, APHIS, Hyattsville, Maryland 20782, (301) 436-8091)

Exotic Ticks on Imported Ostriches

Several ostriches recently imported into the United States from Africa were found infested with exotic ticks. Shipments of infested ostriches reached Texas, Ohio, and Illinois, where both the ostriches and affected premises were treated at appropriate intervals with acaracides approved by the Environmental Protection Agency (EPA). Affected premises, ostriches, and associated animals will remain under surveillance for at least 6 months after the acaracide treatment series is completed, to ensure that no exotic ticks remain alive. A brief report of details follows.

On May 24, 1989, Veterinary Services, Emergency Programs, was notified by the Federal Area Veterinarian in Charge in Texas of unusual ticks collected from recently imported ostriches. The ticks were submitted by Texas A&M University to the Smithsonian Institution, Washington, DC, where they were identified as *Amblyomma gemma* and *Hyalomma* species.

A. gemma is a known carrier of heartwater disease and Nairobi sheep disease, and *Hyalomma* are vectors of East Coast fever, Congo-Crimean hemorrhagic fever in humans, and can cause tick paralysis in domestic animals. These diseases are foreign to the United States.

A. gemma is found mostly on the continent of Africa. It is a three-host tick that infests cattle, camels, sheep, other domestic animals, and humans. *Amblyomma* tick eggs hatch in 7 to 10 weeks. The larvae feed on the host for 4 to 20 days, drop off, and moult within 10 to 120 days. The nymphs then attack a second host, feed for 4 to 20 days, drop off, and moult again in 14 to 30 days. The females attack a third host, engorge for 6 to 25 days, and then remain on the ground for a month before ovipositing. Larvae may live for a year, nymphs 9 months, and adults up to 2 years without feeding. Larvae and nymphs usually attach on the host's head and neck. Older nymphs and adults generally attack the perineum, udder, scrotum, and tail of the host, causing ulcers at the points for attachment.

Hyalomma ticks ("bont-leg-tick" or "strip-leg-tick") are found throughout East Africa. Although cattle and goats are the main hosts, these ticks also attack humans and all species of domestic animals. *Hyalomma* spp. ticks are usually two-host, and, occasionally, three-host parasites. Only the adults are found on larger domestic animals. Earlier stages of the tick are found mostly on small mammals. The site of attachment for the adult ticks is on the host's perineum, udder, scrotum, and tail bush. Tick eggs hatch in 30 days, larvae may molt on or off the same host in 4 to 15 days, and nymphs drop off 3 to 6 weeks after reaching the host as larvae. Female ticks remain on the host 7 days. Larvae can live for a year, nymphs 2 months, and adults 2 years without food. The tick produces ulcers on cattle and often causes lameness in sheep and goats, if attached to the interdigital skin.

Over 800 ostriches have been imported into the United States since January 1, 1989, through USDA-approved commercial bird quarantine stations. The infested group of 50 adult ostriches originated in Zimbabwe in Africa. They arrived at Mundelein, Illinois, on April 2, 1989, and were quarantined for 30 days. The group was released from quarantine on May 2, 1989. Four of these ostriches were sent to Andrews, Texas; 2 to Lamesa, Texas; 3 to Quinlan, Texas; 15 to Turpin, Oklahoma; and 1 to Graysville,

Ohio. All were subsequently quarantined at their respective destinations. Ticks from several birds were forwarded to the National Veterinary Services Laboratories, Ames, Iowa, where they were identified in cooperation with the Smithsonian Institution.

A group of 50 juvenile ostriches was imported through a federally approved quarantine station at Schaumburg, Illinois, January 25, 1989. These had hatched in Tanzania, Africa, from eggs originating in Zimbabwe. On February 25, 1989, 45 of the juvenile ostriches were released from the quarantine facility. Of these, 1 was sent to a zoo in Indiana, 2 to a zoological park in Hawaii, and 42 to an animal dealer in Florida. The dealer stated that 3 more ostriches were added and birds from the group of 45 were sold to various owners in the United States. Dealers' records indicate that 14 were sent to Tampa, Florida; 6 to Live Oak, Florida; 6 to Bushnell, Florida; 8 to Burnsville, Minnesota; 6 to Clayton, Georgia; and 5 to an auction sale in Cape Girardeau, Missouri. Of the five ostriches sold in Missouri, three birds went to Angelton, Texas, and one each to Lawton and Faxon, Oklahoma. All were subsequently quarantined and treated with acaracides. No ticks had been found on the juvenile ostriches up to the time this report was written. (Dr. Adam G. Grow, USDA, APHIS, Hyattsville, Maryland 20782, (301) 436-8073)

Central American and Caribbean Bluetongue Epidemiology

The prospective study of bluetongue (BT) virus infection being conducted in Panama, Costa Rica, Nicaragua, El Salvador, Honduras, Guatemala, Jamaica, Trinidad and Tobago, and Barbados by the Organismo Internacional Regional de Sanidad Agropecuaria, the Interamerican Institute for Cooperation on Agriculture, and the Universities of Florida and Wisconsin, is now entering its fourth year (see 16-3:6-7). Results to date indicate widespread distribution of BT in the Caribbean Basin. However, no clinical disease has been observed in infected animals. Based on typing of antibody responses and over 100 BT virus isolates obtained in the project's Costa Rican laboratory and typed by the U.S. Department of Agriculture (USDA), Agricultural Research Service (ARS) Arthropod-borne Animal Disease Laboratory, the following trends have been observed: in 1986, BT type 1 was widespread in Central America. In 1987, BT types 1 and 6 were isolated in Central America and BT type 12 in the Caribbean. In August 1987, BT type 3 appeared in the southern Caribbean, and by December 1987, BT type 3 was isolated in Central America. In 1988, BT type 3 continued to be isolated in the Caribbean, while BT type 12 was no longer found. BT type 6 continued to dominate in Central America. None of these BT serotypes had previously been isolated in the Western Hemisphere, although earlier serologic studies predicted the occurrence of BT types 1, 6, 12, 14, and 17. Thus, it is unlikely that the recent isolations represent a new epidemiologic development, but rather are observations of an ongoing situation. *Culicoides* midges captured near BT-infected ruminants have included over 25 species; however, over 90 percent have been *C. insignis* . Only isolated specimens of *C. variipennis*, considered the principal vector of BT in the United States, have been found. (Dr. Jane Homan, University of Wisconsin-Madison, (608) 262-1271)

Thermostable Rinderpest Vaccine Development

Current Status. The resurgence of rinderpest (RP) in sub-Saharan Africa in the early 1980's led to renewed interest in RP control and eradication programs. These programs rely primarily on the use of an effective cell culture rinderpest vaccine (CRPV) developed by Plowright in the late 1950's. This vaccine is a modified live

vaccine which must be maintained in a costly "cold chain" during transport and use in the field. Areas directly threatened by RP include many of the less-developed countries; cold chain requirements such as ice machines, electrical generators, refrigeration equipment, and fuel are a severe economic and logistic burden on their national veterinary services.

The need for a thermostable vaccine was recognized, and several research groups have been working toward this end. The approaches have included the development of new thermoresistant vaccine strains by thermal selection, the development of a vaccinia-vectored RP vaccine, and the development of thermostable conventional Vero cell-adapted RP vaccine (VRPV) through improved lyophilization and chemical stabilization techniques.

Thermoresistant Strains. The approach to a thermoresistant strain relies either on growing the virus at elevated temperatures (i.e., 42 °C) or subjecting the virus to high temperature pulses (i.e., 56 °C), and growing out surviving particles. These methods have been applied to a closely related Paramyxovirus, Newcastle disease virus, and were reported to have resulted in a thermostable food pellet vaccine for poultry. This approach has been pursued for RP vaccines both at the Foreign Animal Disease Diagnostic Laboratory (FADDL) on Plum Island and at the Institut d'Elevage et de Medecine Veterinaire des Pays Tropicaux in Paris. Although promising strains showing improved thermostability have resulted, none has received the extensive animal immunogenicity and safety testing required in development of vaccine seed strains.

Vaccinia-vectored RP Vaccines. Vaccinia is noted for its stability and was an important component of the successful World Health Organization (WHO) campaign for the global eradication of smallpox. Several recombinant vaccines against a variety of diseases have been produced which rely on vaccinia as an expression system for immunogenic proteins. The essential method is to isolate the mRNA coding for an immunogenic protein and produce cDNA clones to these sequences. These cDNA inserts are introduced into the vaccinia virus by a complex process, ultimately relying on homologous recombination. This has been done for the two important immunogenic proteins of RPV, the hemagglutinin (HA) and fusion (F) protein. Yilma *et al.* (Science, 242:1058-1061, 1988) have protected cattle against challenge with the virulent RBOK strain using vaccinia recombinants expressing either the HA or F protein. Recently, Barrett *et al.* (Virology 170:11-18, 1989) reported protecting rabbits and cattle with a vaccinia recombinant expressing the F protein.

Although the stability and efficacy of these recombinants is very promising, questions remain regarding the safety of vaccinia-vectored vaccines. Vaccinia, including the WR-attenuated strain used in the WHO smallpox eradication campaign, is known to be capable of producing a life-threatening infection in man. The high incidence of immunosuppressive diseases, such as AIDS, in developing countries increases the concern associated with use of vaccinia-vectored vaccines. The essential question is, "Are the human health risks associated with the use of vaccinia, which were reasonable in relation to the control of smallpox, appropriate to the control of an animal disease?"

Barrett (ibid. 1989) makes several suggestions toward improving the safety of recombinant RP vaccines. These include the use of further attenuated strains of vaccinia or other pox virus species that are nonpathogenic for man, such as fowl pox or members of the Capripox virus genus. Further attenuation of vaccinia can be achieved by deleting the vaccinia growth factor gene or introducing the gene for interleukin 2.

Vero Cell-Adapted RP Vaccine. FADDL scientists collaborating with Tufts University School of Veterinary Medicine have used conventional methods to develop a thermostable RP vaccine. The vaccine is based on the Muguga modification of the Kabete O strain (Plowright strain) adapted to Vero cells. Comparisons were made of several promising rinderpest, canine distemper, and measles vaccine stabilizers to determine the most efficacious formulation. Surprisingly, a lactalbumin hydrolysate and sucrose RP stabilizer used by Plowright provided the best stabilization and outperformed preparations based on sorbitol and hydrolyzed gelatin used in thermostable measles vaccines. Improvements to the lyophilization cycle resulted in a vaccine which maintained the Office International des Epizooties (OIE) minimum required dose for over 245 days at 37 °C. Based on the OIE norms for RP vaccine storage "expiry periods," VRPV has adequate stability for storage at ambient temperatures for more than 30 days (Mariner *et al.*, Vet. Micro., in press). Thus, VRPV combines the safety and efficacy of the Plowright vaccine with sufficient thermostability for use without a cold chain for at least 30 days.

The Vero cell production system replaces the primary cell production system with a continuous cell line. Vero cells have the advantage of yielding consistently high titer RP virus harvests with less risk of adventitious viral contaminants. In addition, Vero cells eliminate some of the cell supply problems associated with the primary bovine kidney cells used in the production of CRPV.

The thermostable VRPV has been successfully field tested under severe environmental conditions in the African Sahel (Mariner, *et al.*, Vet. Micro., submitted for publication). In this test, the vaccine was stored at ambient temperatures for over 30 days in Niamey, Niger, and then used to vaccinate 144 seronegative cattle. The vaccine retained more than adequate titer and resulted in 98 percent seroconversion among the vaccinates. Currently, Niger is producing and evaluating production scale lots of VRPV for use in the next RP campaign (1990). The field use of the thermostable vaccine without a cold chain will be limited to one or two departments in the first year. In addition, representatives of the Pan-African Rinderpest Campaign (PARC) have expressed interest in adopting the production techniques on a regional basis. (Dr. Jeffrey Mariner, USDA, APHIS, Foreign Animal Disease Diagnostic Laboratory, (516) 323-2500, ext. 283)

Focus On—Akabane Disease

Akabane virus causes congenital arthrogryposis and hydranencephaly (A-H) in the bovine, caprine, or ovine fetus after infection *in utero*. Less frequently, the virus causes polioencephalomyelitis. Congenital A-H syndrome results from infection of the fetus during the first trimester of pregnancy when Akabane virus and possibly other antigenically related arboviruses are transmitted by mosquitoes or biting midges (*Culicoides* spp.) to the pregnant dam. Fetal infection causes abortions, stillbirths, premature births, mummified fetuses, and various dysfunctions or deformities of fetuses or liveborn calves, kids, or lambs. Adult ruminants are not clinically affected except for the dystocia caused by the fetal deformities.

Etiologic Agents

Congenital A-H syndrome is caused by teratogenic Simbu serogroup viruses of the family *Bunyaviridae*. Currently, the International Catalogue of Arboviruses, Including Certain Other Viruses of Vertebrates (Karabatsos, 1985), lists 21 antigenically related Simbu serogroup viruses. Ten of these viruses and Facey's Paddock virus, an Australian virus not yet registered in the Catalogue, have been isolated from livestock or vectors: Sabo, Sango, Shamonda, and Shuni in Nigeria; Douglas and Peaton in Australia; Akabane in Australia, Israel, Japan, and Kenya; Tinaroo in Australia and Papua New Guinea; Thimiri in Australia, Egypt, and India; and Sathuperi in Africa and India. In addition to Akabane virus, Aino (Samford), Douglas, Peaton, and Tinaroo viruses have been implicated as causative agents of congenital A-H syndrome. All of these viruses are exotic to the United States.

Viruses of the Simbu group are characterized by a single-stranded ribonucleic acid genome, comprised of three segments (L, which codes for the large protein believed to have transcriptase activity; M, which codes for the glycoproteins; and S, which codes for the nucleocapsid and another nonstructural protein), with a total molecular weight of 6-7 million daltons. By using polyacrylamide gel electrophoresis, these viruses have been shown to contain at least four identifiable polypeptides; L, G_1, G_2, and N. It has been suggested that the G surface glycoprotein is partially responsible for the fetal tissue tropism of Akabane virus. The nucleoprotein carries the group-specific antigens; and the envelope glycoproteins contain the type-specific antigens involved in neutralizing and hemagglutinin-inhibiting antibody responses. The virions are spherical, enveloped particles with helical symmetry, and are 90-100 nm in diameter. The virions are inactivated by lipid solvents and deoxycholate. Viral replication occurs in the cytoplasm of infected cells such as hamster lung cells (HmLu-1) and monkey kidney cells (CV-1) in which these viruses will produce plaques, induce cytopathic changes, and replicate to high titer ($>10^{7.5}$ pfu/ml). Neutralization tests are strain-specific, while immunoprecipitation and immunofluorescence tests are more broadly cross-reactive.

History and Geographic Distribution

Congenital A-H syndrome was first described in calves in Australia in 1954. Although the seasonal occurrence of the syndrome was recognized and the infectious nature was suspected, no etiologic agent was isolated. Akabane virus is enzootic in northern Australia, with occasional epizootic incursions into southern Australia. In Australia, the distribution of antibodies to Akabane virus closely follows the distribution of *Culicoides (C.) brevitarsis*, or to the north and east of the so-called "brevitarsis line." The areas

involved include the north of Western Australia, most of the Northern Territory and Queensland, and the northern Tablelands and coastal parts of New South Wales. While periodic outbreaks of congenital A-H syndrome had been reported since 1949, during the summer, fall, and early winter months from 1972 to 1975, an epizootic of abortions and stillborn and deformed calves with congenital A-H syndrome occurred in central and western Japan. Akabane virus, which had been isolated previously from *Aedes* and *Culex* spp. mosquitoes, was serologically incriminated in the outbreaks and was isolated later from naturally infected fetuses. Subsequently, Akabane virus was used to reproduce the congenital A-H syndrome in the fetuses of experimentally infected pregnant cows, ewes, and goats.

Congenital A-H syndrome attributable to Akabane or other serologically related, teratogenic Simbu group viruses has also been reported in Argentina, Cyprus, Turkey, Kenya, South Africa, Zimbabwe, and other African countries; Israel and other countries of the Middle East; and southeast Asia, including the countries of Bandung, Indonesia, Korea, Malaysia, The Philippines, Taiwan, Thailand, and Vietnam. Akabane and related viruses have a wide distribution among cattle and other domestic animals in tropical and temperate areas of the world. However, congenital A-H syndrome has a relatively limited geographic distribution.

Akabane Virus Hosts

Infection of pregnant adult cattle, sheep, and goats causes no overt clinical signs, but fever and viremia occur. Transplacental passage of the teratogenic virus to the bovine, caprine, or ovine fetus can occur at the time of infection of the pregnant adult, but the clinical consequences may not be manifested until later in pregnancy. There have been no reports of congenital A-H syndrome related to Simbu group virus infections in the fetuses of wild ruminants or other mammals, although antibodies have been detected or virus isolated from the following: Aino virus in buffalo and horses; Akabane virus in buffalo, camels, deer, dogs, horses, and monkeys; Douglas virus in buffalo, deer, and horses; Peaton virus in buffalo, deer, horses, and pigs; and Tinaroo virus in buffalo and horses.

Transmission and Epizootiology

Akabane and the other Simbu group viruses are arthropod transmitted. There is no indication of methods of transmission other than by insect vectors. The occurrence of congenital A-H syndrome is seasonal and geographic.

The pathogenesis of the Simbu serogroup viruses for the ovine fetus appears to depend upon the ability of the virus to cross the placental junction at the proper gestation age. The resulting pathologic changes determine the extent of developmental abnormalities. It has been suggested that only the Simbu group virus isolates with electrophoretic protein profiles similar to Akabane virus may be teratogenic; this could explain the limited geographical distribution of the congenital A-H syndrome.

The location and timing of the infection of the fetus during the first trimester of pregnancy is consistent with the seasonality of transmission by hematophagous insects. In southeastern Australia, where the suspected vector species, *C. brevitarsis*, is a bundant from October to March; the virus transmission season is October to November; whereas in northern Australia, the vector season is May to September and the virus transmission occurs from June to October. Akabane virus has been isolated

from *Aedes vexans* and *Culex* (Cx.) *tritaeniorhynchus* mosquitoes and *C. oxystoma* biting midges in Japan; *Anopheles funestus* mosquitoes in Kenya; a mixed pool of *Culicoides* in South Africa; and C. *brevitarsis* and *C. wadai* biting midges in Australia. In addition, Aino virus has been isolated from *Cx. tritaeniorhynchus*, a mixed pool of *Cx. pipiens/pseudovishnui*, and *C. brevitarsis*; and Douglas, Peaton, and Tinaroo viruses have been isolated from *C. brevitarsis*. Evidence of biological transmission by these species is still lacking, although epizootiologic evidence incriminates them. In Australia, *C. brevitarsis* and, in Japan, *C. oxystoma* are believed to be the primary vectors of Akabane virus. Of these suspected vectors, only *Cx. pipiens* occurs in the United States. The question of possible transmission by other North American mosquitoes or biting gnats, such as *C. variipennis* and *C. insignis*, remains unanswered.

Signs and Lesions

Clinically, the congenital A-H syndrome is seasonally manifested as a sporadic epizootic of abortions, followed some months later by stillbirths, premature births, and deformed or anomalous bovine, caprine, or ovine fetuses or neonates. The pregnant dam experiences fever at the time of infection with the virus. If infection occurs during the first trimester of pregnancy, fetal damage occurs *in utero.* Dystocia may occur in the dam at parturition due to the types of lesions produced in the fetus. Badly deformed fetuses are usually dead at birth and the limbs are locked in the flexed or extended position. Most live neonates have degenerated central nervous systems (CNS) and muscles that prevent the animal from standing or suckling. Calves born with hydranencephaly may survive for months if they are hand-reared, but they will not thrive. Torticollis, cervical scoliosis, brachygnathism, and kyphosis may coexist with arthrogryposis. Lesions in the CNS may be expressed clinically as blindness, nystagmus, deafness, dullness, slow suckling or dysphagia, paralysis, and ataxia. Lesions and death may be observed in virtually all fetuses or neonates of approximately the same gestational age.

In the dam, fever and viremia generally occur within 1-6 days after infection. Viremia lasts 3-5 days and neutralizing antibodies are detectable in ewes after the fourth day of infection. Fetal infection is not apparent until much later in pregnancy. Timing of the infection relative to the gestational stage is critical to the development of fetal clinical effects. In pregnant ewes, the critical period for production of fetal abnormalities has been shown to be from 30 to 53 days of gestation. In pregnant cows, infection from 62 to 96 days of gestation and, in pregnant goats, infection at approximately 40 days of gestation produced fetal lesions. In field studies, it was shown that 17.2 percent of calves born to cows infected with Akabane virus between 76 and 224 days of gestation were abnormal.

Fetal lesions are associated with muscular and CNS damage. Arthrogryposis is the most frequently observed clinical sign of infection. Affected joints cannot be straightened even by application of force, due to ankylosis of the joint in the flexed or extended position. Subcutaneous petechial and ecchymotic hemorrhages may occur on the fetus or the placenta, and white, turbid spots may be observed on the amnion. Shallow erosions may occur on the external nares and muzzle, and between the distal digits. Hypoplasia of the lungs and skeletal muscles, fibrinous polyarticular synovitis, fibrinous navel infection, ophthalmia, cataracts, and presternal steatosis also occur. Within the CNS, hydranencephaly, hydocephalus, agenesis of the brain,

micrencephaly, porencephaly, and cerebellar cavitation, fibrinous leptomeningitis, fibrinous ependymitis, and agenesis or hypoplasia of the spinal cord are variously reported.

Microscopically, a mild-to-moderate neurogenic skeletal muscle atrophy and polymyositis are observed. In the CNS, lesions include encephalomyelitis, secondary demyelinization of the spinal cord and motor spinal nerves, micrencephaly, cerebellar cavitation, and cystic areas and malacia, especially in the cerebrum. There is also generalized edema, subependymal gliosis, perivascular cuffing, neuronophagia, and mineralized plaques in the brains.

Diagnosis

A field diagnosis of congenital A-H syndrome can be made on the basis of the clinical picture, gross pathologic lesions, and epidemiology. The sudden onset of aborted, mummified, premature, or stillborn fetuses with arthrogryposis and hydranencephaly should be suggestive. The dam will have had no clinical history of illness. A retrospective study will likely show that the first trimester of pregnancy occurred during a time of biting insect flight activity.

Although virus isolation at term is unlikely, virus isolation should be attempted from the placenta, fetal membranes and fluids, fetal muscle, or fetal nervous tissue. Akabane virus and the other Simbu group viruses are easily isolated by the intracranial inoculation of suckling mice and in various cell cultures. Virus isolates are identified by immunofluorescence staining procedures which also may be used to identify viral antigens in fetal tissues.

In the absence of virus isolations, a serologic diagnosis may be made by demonstrating neutralizing antibodies in precolostral or fetal serum samples. In adult animals, seroconversion or a demonstrable rise in antibody titer would indicate infection. The presence of specific antibodies in precolostral serum of the affected neonates and in the serum of the dam has proved most useful for serologic evidence of *in utero* Akabane virus infection. A serum neutralization test, a hemagglutinin-inhibition test, a complement fixation test, a hemolysis-inhibition test, an immunoprecipitation test, and an immunofluorescence test are available for detecting and assaying antibodies.

A variety of nutritional, toxic, and infectious diseases will produce fetal wastage and deformities. Rift Valley fever, bovine viral diarrhea, bluetongue, sporadic bovine abortion, Wesselsbron virus infection, and epizootic ovine abortion would present the greatest difficulties in a differential diagnosis. Recently, arthrogryposis and CNS malformations in neonatal lambs were associated with Cache Valley (CV) virus infection in the United States. CV virus is a bunyavirus but it is not a Simbu group virus.

Akabane Control

The arboviral agents that cause congenital A-H syndrome can be controlled by vector control techniques typically recommended for other arbovirus diseases. Vector control depends upon disruption of breeding sites, reduction of vector populations with pesticides or biocontrol techniques, and protection of host animals from feeding of the vectors by using repellents or housing during the feeding hours of vector species.

Female ruminants may be protected by vaccination before breeding. Akabane vaccine is not available in the United States. A formalin-inactivated, aluminum phosphate gel-absorbed vaccine and an attenuated vaccine have been developed in Japan for Akabane virus. In Australia, a beta-propiolactone inactivated vaccine in aluminum phosphate gel adjuvant was found to be highly immunogenic and protected 98.9 percent of vaccinated cows. Immunizing agents for other Simbu group viruses are not currently available. Research likely will not begin until the true pathogenic potential of these viruses has been defined. Genetic cloning and artificial expression of the M gene or the two glycoproteins which are the major surface antigens is currently being done by Australian scientists. These products of molecular biologic research offer promises for genetically engineered vaccines and future control.

Akabane References for Further Reading

Beveridge, W. I. B., 1986. Akabane disease. *In:* Animal Health in Australia. Viral Diseases of Farm Livestock, Arbovirus Diseases. Australia Government Publishing Service, Canberra, 1:3-10.

Della-Porta, A. J., and Gard, G. P., 1981. Akabane disease. Report from the SCA-Animal Health Committee published by the Australian Bureau of Animal Health,1-27.

Inaba, Y., and Matumoto, M., 1981. Congenital arthrogryposis-hydranencephaly syndrome. *In:* Virus Disease of Food Animals. A World Geography of Epidemiology and Control. Disease Monographs. E. P. J. Gibbs, ed. Academic Press, New York, 2:653-671.

Parsonson, I. M., Della-Porta, A. J. , and Snowdon, W. A., 1981. Developmental Disorders of the Fetus in Some Arthropod-borne Virus Infections. Amer. J. Trop. Med. and Hyg. 30:660-673.

St. George, T. D., and Standfast, H. A., 1988. Simbu Group Viruses with Teratogenic Potential. *In*: The Arboviruses: Epidemiology and Ecology. T. P. Monath, ed. CRC Press, Boca Raton, FL, 4:145-166.

(Thomas E. Walton, USDA, ARS, P. O. Box 3965, University Station, Laramie, WY 82071-3965)

Questions about the FAD Report may be sent to:

Dr. Edwin I. Pilchard, Editor
USDA, APHIS, VS
Room 741, Federal Building
6505 Belcrest Road
Hyattsville, MD 20872

Thirty days before moving, send address change and, if possible, mailing label from latest issue to:

Information Management Branch
APHIS-USDA
Room G-100, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

☆ U.S.G.P.O.: 1989- 717-013/04485